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STUDIES IN THE DECIDUA
AND
Retained Membranes of the Ovum.

VULVAR OEDEMA
Complicating Pregnancy.

VERSION BEFORE LABOR
IN
MAL-POSITIONS OF THE FOETUS.

BY
EDWARD A. AYERS, M. D.

Lecturer in Obstetrics, New York Polyclinic, N. Y.



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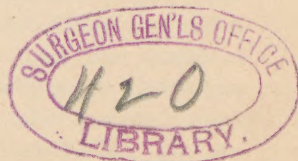
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STUDIES IN THE DECIDUA AND RETAINED MEMBRANES OF THE OVUM.

BY EDWARD A. AYERS, M.D.,
Lecturer on Obstetrics in the New York Polyclinic.

IN my investigations with the microscope of retained membranes, I desired to note some of the changes leading to death of the ovum, its retention, and consequent alterations. The main points I claim to have observed are : First, transformation of the epithelia in the utricular glands in some portions of the decidua into decidual tissue ; second, an inflammatory process in the avascular amnion, which I term amnionitis ; and third, decidual hyperplasia.

CASE I. *Retained Decidua, showing Transformation of Epithelia in the Utricular Glands into Decidual Tissue.*—The metamorphosis here observed, involving as it does the mooted question of the limitations in epithelial tissue transformations, deserves a minute and conscientious description.

In a normal uterine mucous membrane we find, previous to menstruation, the base embedded in a muscular stratum of great thickness, as compared with the same in other organs. Previous to the first menstruation, the middle stratum or corium is about one millimetre (one-twenty-fifth inch) thick.

After the first menstruation it becomes permanently about two millimetres thick. It is composed of loose vascular connective tissue containing few fibres but much structureless interfibrillar substance, and has scattered through it very numerous, round, fusiform, or irregularly shaped granular nucleated cells. The inner surface is lined by a single layer of columnar ciliated epithelia. The numerous tubules or utricular glands which open into the uterine cavity pierce this layer at different angles ; not as

drawn by Weber, and since so often copied into various text-books, where they appear as straight parallel tubes entering the uterine cavity at right angles, but usually at an angle of about forty-five degrees, though the most conspicuous feature is the irregularity of their arrangement. They branch as they go deeper into the mucosa, where they are very irregularly distributed. The walls of the utricular glands are composed of a very delicate fibrillar tissue, arranged concentrically, and attached externally to the interglandular connective tissue. Internally is a single layer of ciliated columnar epithelia, unenucleated, continuous, and apparently indential in structure with the columnar epithelia lining the free surface of the uterine cavity. The epithelia in the glands are attached to one another by a transparent intercellular cement substance. They are generally uniform in shape and size.

Preliminary to the menstrual flow, there is a marked growth in the interglandular connective tissue, the mucous membrane becoming from three to six millimetres thick; the lymph-channels widen and the glands become longer, wider, and more patulous; the blood-vessels become over-distended and either extravasation or diapedesis follows, probably extravasation. So far authorities agree. According to Kundrat and Englemann, fatty degeneration of the glandular epithelia, the inter-glandular blood vessels, and the connective-tissue corpuscles follows the superficial desquamation of tissue. This fatty degeneration is limited by them to the superficial layers of the mucosa, the new mucous membrane arising from the intact deeper layers. Leopold opposes the theory of fatty degeneration, while Williams holds that the entire mucous membrane is removed at each menstruation, the new membrane being regenerated from the muscular coat.

As against Williams' view, Klebs and Heller hold that the epithelium of mucous membranes is reproduced only so

long as the deeper layer or matrix of epithelial cells is intact, and that when these are destroyed the cells at the margin fill in the gap, and if the marginal epithelia are also wanting, the space becomes occupied by cicatricial tissue.* In short, it is a general law that like tissues produce like tissues, or the space is occupied by cicatricial connective tissue.

Self-regeneration of striped and non-striped muscular tissue has been shown, through the researches of Kraske, Erbkam, and Heidelberg, † to occur to a limited extent, but not the regeneration of mucous from muscular tissues. If now the epithelia of the utricular glands be considered as a tissue distinct from decidual tissue, in the sense of the general law of regeneration just given, it follows that that law must have exceptions, or that I have incorrectly interpreted the observations I am about to describe. On the other hand, decidual tissue and epithelial tissue may not be distinct in the sense of that general law, in which case no conflict will be resultant. Passing now to special observations of my own, the membranes I examined were from specimens obtained by Dr. Boldt, who curetted the uterine cavity of a girl, twenty years of age, she having recently aborted. The decidua was of about two months' growth. The specimens I examined were stained in ammoniacal carmine and mounted in glycerine. With low powers of the microscope the presence of utricular glands is readily recognized, characterized by the lining of the columnar ciliated epithelia and a distinct calibre, both in transverse and oblique sections. A large number of sinuous venous cavities is likewise present, varying in size and mostly filled with disintegrating blood, composed of granules of a greenish color, owing to their preservation in a chromic acid solution. As some of the smaller veins contain un-

* Landois and Stirling, p. 402.

† Ibid., p. 403.

changed blood corpuscles, it is reasonable to believe that the circulation in the veins holding disintegrated blood had ceased some time before the removal of the membrane, and the changes incident in thrombosis occurred.

Between the glands and the venous cavities is a well-pronounced decidual tissue of the myxomatous type, traversed by a moderate number of capillaries, most of which contain unchanged blood corpuscles. The myxomatous net-work of the decidual tissue varies in size in different places, being small around the veins and wide in the portions between the veins at the periphery of the nodule (see Fig. 1). The "decidual cells" of Friedländer or Kolliker, termed by Hirst "enormously enlarged young connective tissue cells," occupy the greater portion of the interglandular space.

The utricular glands seen in Fig. 1 all show more or less alteration from a normal outline. They are all much enlarged as compared with those in the endometrium previous to impregnation.

In those portions of the glands where no structural change has taken place the columnar epithelia show the characteristic features of form and arrangement, and are possessed of single nuclei of large granular appearance. The cell substance has a granular appearance finer than that in the nuclei. The cilia are not preserved, having been lost in the preparation of the specimen. Surrounding the epithelia is a circular band of delicate fibrillar tissue, termed anhistous or formless by Velpeau, which is continuous in the unchanged portions of the glands, being interrupted or lost in other parts. The calibre of the glands is changed as the epithelia have become altered. In the altered portions of the glands the changes noted are as follows: In the columnar epithelia a varying degree of change in form and construction is seen, so that they are no longer "columnar" but most irregular in shape and

have become more or less divided and subdivided. The unusual arrangement of single nuclei has disappeared, but each subdivision of the cells now holds a nuclear body



FIG. 1.—*A*, Utricular gland; *B*, decidual tissue; *C*, empty vein; *D*, vein filled with disintegrated blood; *E*, *E*, capillaries of decidual tissue.

more homogeneous and refractive than the original nucleus. Where only a limited part of the epithelial ring is involved in this change the free inner margin of the cells extends or bulges and occupies a portion of the calibre of the tube, thus rendering the cavity sinuous and irregular. At the same time the outer boundary line, the circular anhistous tissue, is lost, and the altered epithelia appear to be morphologically similar and continuous with the adjacent de-

cidual tissue. Where the tissue change has involved a majority of the epithelia, the calibre of the gland is invariably reduced, as if by compression. In the highest degree of this change only a vestige of the calibre is left, which then disappears, being filled with decidual tissue. These changes obviously lead to a destruction, as such, of the utricular glands, and deserve our most careful attention. With high powers of the microscope the gradual changes of the utricular glands, particularly the epithelia, can be traced to their complete disappearance through metamorphosis. In Fig. 2 is shown a utricular gland magnified five hundred diameters. In a considerable portion of the tubule the columnar epithelia are essentially unchanged; they retain their usual form, are possessed of single nuclei, showing the coarse granules with finer granules entering into the construction of the "cell-substance." The lateral union of the epithelia shows a double line with the interspace filled with the so-called "intercellular cement substance." At their base is the anhistous tissue which forms the outer boundary of the tubule. In the remaining portion of the tubule a generative change has begun in the epithelia; they have lost their regularity of form, have become divided and subdivided. The intercellular structure is in a transitional stage, being reduced to a single line of division, and the single nuclei have given place to many nuclei of varying appearance, size, and location.

There is now to be seen a multinuclear protoplasmic mass, representing an increase in tissue, as is shown by its encroachment on the calibre of the tubule. The increase in size, and especially in number, of the granules or new nuclear bodies in place of the previous nuclei is marked. These bodies are recognized as "mother-cells," the term for endogenous new formation in epithelia. This latter process is understood to mean a splitting up, or better, a division of, and increase in, living matter in the nuclei of

epithelia—that is, karyokinesis. This process, in addition to the views of Schumann, is now, owing to the more recent researches of Bütschli, Flemming, Van Beneden, Waldeyer, and others, recognized as one of the most important biological discoveries of the past twenty-five years, particularly on account of its relation to fertilization of the ovum. This process invariably occurs in inflammatory and other changes in epithelia, leading either to a new formation, or growth, or a reduction into a medullary or embryonal condition of

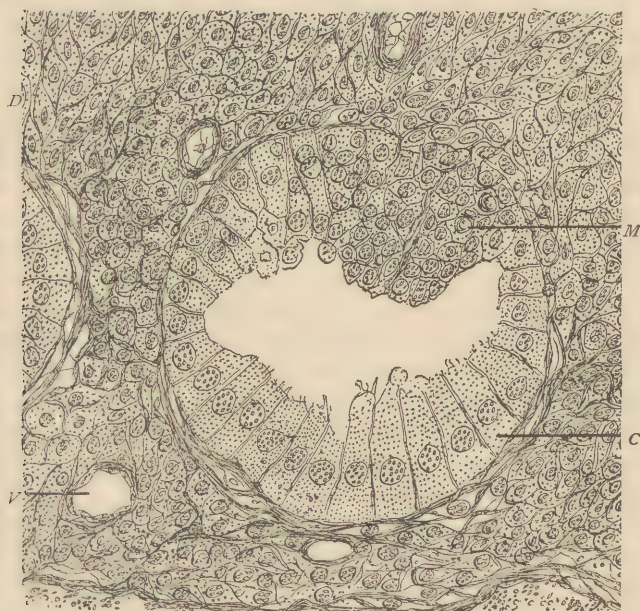


FIG. 2.—*C*, Columnar epithelium of utricular gland, unchanged; *M*, epithelium transformed to medullary tissue; *D*, myxomatous decidual tissue; *V*, capillary blood-vessel in transverse section.

the epithelial tissues. Not infrequently light, so-called plasmatic spaces surround the newly-formed granules or

nuclei. Observing further we see the second stage, the so-called medullary or embryonal, in which the epithelia break up into nucleated protoplasmic bodies, varying in size, and separated from one another by light lines of an intercellular cement substance. This cellular division is seen to be most complete in the more central parts of the changing tissues, and only partial at the junction with unchanged epithelia. The intercellular cement substance shows in its construction a certain amount of living matter, and by a consolidation produces the myxomatous reticulum, while the original medullary bodies have grown up into nucleated so-called "decidual cells," now considered as protoplasmic masses filling the meshes of the myxomatous reticulum. I have mentioned that in places at the periphery of the tubule, where the transformation of the epithelia is going on, the bordering line of the utricular gland is lost, and the medullary tissue sprung from the epithelia blends without demarcation with the adjacent decidual tissue, as is shown in Fig. 2.

Decidual tissue is generally admitted to be myxomatous connective tissue. Most modern microscopists limit the variability of epithelial tissue to such a degree that only epithelial formation can be derived from it. It is clearly shown in my specimens that there is a transformation of the glandular epithelia through an intervening stage of embryonal or medullary into decidual tissue.

The objection may possibly be raised that I have misinterpreted the changes observed; that the transformation was one of proliferation of the extra-glandular decidual tissue and its substitution for the epithelia. The alteration in the epithelia was not a degenerative or destructive process, no desquamation had taken place. The various grades of endogenous new formation in the epithelia can be clearly seen. Nor does the circular anhistous tissue, which surrounds the glands and stands as a barrier between the

epithelia and the outer decidua tissue, disappear until the alteration in the glandular epithelia is well advanced.

On the other hand, it cannot be claimed that the specimens here described permit the claim that all decidua tissue arises from the glandular epithelia. Ercolani* holds that the human decidua is, for the most part, a product of exudation from the utricular glands. The "epichoreal" decidua (reflexa) has no utricular glands.† They are present in both the "placental" decidua (serotina) and "uterine" decidua (vera), but remain open longer in the former than in the latter, where they are finally found only in the deeper layers.‡ It is probable, therefore, that the specimens herein described are from the decidua uterina or vera.

How do the statements here made harmonize with our present knowledge of tissue regeneration?

Without attempting an extended answer to this question, I wish to suggest a few points. Quoting from Professor Marcy : || "Except as viewed by Ercolani, the opinions which have been entertained concerning the origin of the cells which enter so largely into the formation of the maternal portion of the placenta have been exceedingly vague and uncertain. Professor Turner, although concisely affirming, with Owen, that without decidua there is no formation of placenta, does not touch the important question of the origin of the decidua. Professor Kölliker confines himself to the remark that the decidua is a transformation of the uterine mucous membrane, and not a new membrane or the product of an exudation, as was once believed. It may be observed, however, that Professor Kölliker does not attempt to show which are the elements in the mucous

* Ercolani : Utricular Glands of the Uterus (Marcy), pp. 24 and 134.

† Landois and Stirling, p. 883.

‡ Hirst's American System of Obstetrics, vol. 1., p. 257.

|| Placental Development ; Transactions of North American International Medical Congress, vol. ii.

membrane that compose the decidua, nor by what means they are transformed, although such an investigation should be of the highest interest to him, since the glandular follicles of new formation, as demonstrated by Professor Ercolani, and largely confirmed by Turner, were only declared by him to be tumefactions of the pre-existing uterine mucous membrane, formed during pregnancy and disappearing after delivery."

As a rule, the glandular epithelia of mucous membranes, derived from the internal layer of the blastoderm, perform their chief physiological function by entering in, following desquamation, as an essential part of the secretion poured out by the glands, as in the lactiferous glands, for example. In the utricular glands, however, the secretion during non-pregnant periods is an indifferent mucus having no nutritive function. I agree with Dr. Putnam-Jacobi,* that "since it is from the epithelium (the original hypoblast elements) that the serotina or 'glandular organ' of the placenta is formed, and since it is the epithelium from which the entire mucosa is regenerated, both after parturition and after the less severe exfoliation of menstruation, it is evident that the epithelium is of great importance among the elements of the endometrium. The epithelium of the glands is simply a prolongation of that lining the cavity in a series of convolutions. From this point of view these glands may be considered simply as folds of epithelium, convoluted as is the surface of the brain, by the usual organic device for multiplying extent of surface within a given space." Comparative anatomy and physiology present facts that favor this view. For example, the hare has no utricular glands—simply crypts lined by involutions of the surface epithelium.

A comparison of the conditions present in extra-uterine tubal pregnancies with those of intra-uterine, gives striking

* Mary Putnam-Jacobi; Studies in Endometritis, American Journal of Obstetrics, March, 1885, p. 270.

evidence in favor of the views here expressed. Here we can eliminate the "different species" equation, and obtain some knowledge of essentials in gestation. The Fallopian



FIG. 3.—Mola Carnosa. Natural size.

tubes have no glands, but an extensive surface of involuted mucous membrane resting upon a muscular base. The free surface of the mucous membrane, which if stretched out would be very extensive compared with the space it occupies, is lined with ciliated columnar epithelium, differing in no way apparently from that lining the endometrium, except that the cilia move in the opposite direction, each moving in the direction of the cornua. Notwithstanding the absence of any glandular arrangement in the tubes, a successful gestation is quite possible. Nor is the customary premature termination of gestation by hemorrhage or rupture apparently due to the inability of the mucous membrane to perform its share in the unusual gestation.

The utricular glands cannot be said to be essential, then, as glands whose function is to supply a secretion or excretion, and doubt may fairly be cast upon the theory of Ercolani, that the decidual tissue is for the most part a "product of exudation from the utricular glands."

CASE II. *Fleshy Mole ; Decidual Hyperplasia and Amnionitis*.—The specimen obtained in this case, which was presented to me by Dr. McGillicuddy, is of unusual occurrence and interest, and justifies some special attention clinically as well as microscopically.

Mrs. M——, aged thirty-four, married five years, had two children and afterward two miscarriages. Her first miscarriage occurred in February, 1888, at two months'

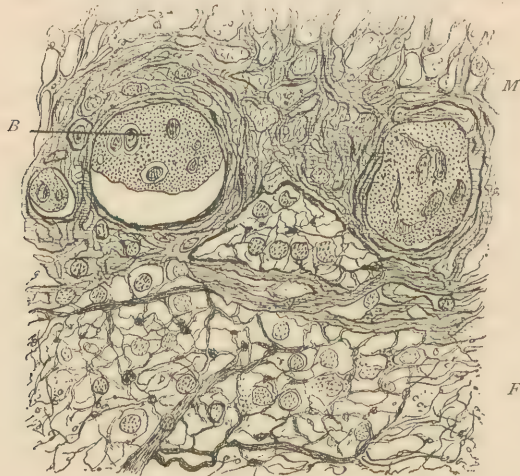


FIG. 4.—*B*, Protoplasmic buds ; *F*, coagulated fibrine ; *M*, hyperplastic myxomatous reticulum of decidua.

gestation. Menstruation was resumed the following 12th of June. Impregnation followed. Morning sickness developed during the latter part of July. August 10th, a uterine hemorrhage ensued, supposed to have been caused

by lifting a heavy trunk. The hemorrhage lasted three days. No pain was experienced. Hemorrhages followed on September 9th and November 8th. The latter lasted seven days, when, preceded by severe pains, the specimen shown in Fig. 3 was passed from the uterus. A segment has been removed to show the cavity, in which is seen, in natural size, a fœtus of four weeks' development, attached to the wall of the ovum by the allantois.

From the history and the stage of development shown by the fœtus, its death must have occurred in August with the first hemorrhage. It is somewhat remarkable that during the three months in which it was retained, no absorption of its tissues took place. The cavity in which the fœtus floated was lined with a smooth, but nodular, corrugated membrane—the amnion. The dense tissue composing the wall or body of the mass was traversed with clotted blood; the latter, indeed, representing the greater part of the mass. Nevertheless, it is more properly termed a “mola carnea” than “mola sanguinea.”

Although the embryo died at the end of four weeks

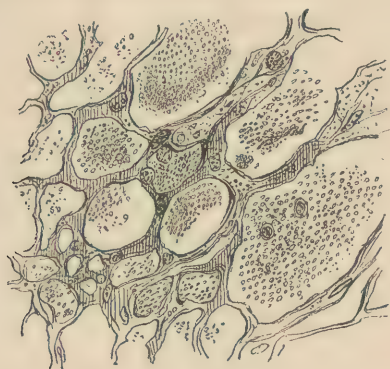


FIG. 5.—Diffuse Hyperplasia of Decidual Endometrium. $\times 500$.

yet the placental tissue continued to develop, pathologically, up to the time it was expelled.

The mounted specimens prepared from it, if viewed with low powers, show the main mass consisting of blood-clot; in some places with masses of red blood-corpuscles, in others with masses of coagulated fibrin. Imbedded in these masses are seen remnants of villositities, cut in all directions, mostly compressed, and with but scanty vestiges of blood-vessels also compressed and collapsed.

The placental tissue is preserved in but few places, and in a peculiarly altered, condensed condition. The periphery is composed of the amnion-producing nodular bulgings from the adjacent decidual tissue, and greatly varying in thickness in different parts of the specimen.

Some portions of the amnion are very little changed in thickness, and in this situation are characterized by a lining of short columnar epithelia toward the cavity holding the amnion.

Other portions are about twelve times as thick, and thus likewise cause irregularly corrugated and nodular protrusions. Such places show a variety of protoplasmic bodies, either globular, oblong or spindled, imbedded in a dense striated tissue, obviously the outcome of a chronic formative inflammation. As I was able to trace the different stages of the inflammatory process from its first stage in the acute form up to a marked increase of the amniotic tissue, I am induced to describe a, so far as I know, hitherto unknown pathological process, for which I propose the term, amnionitis.

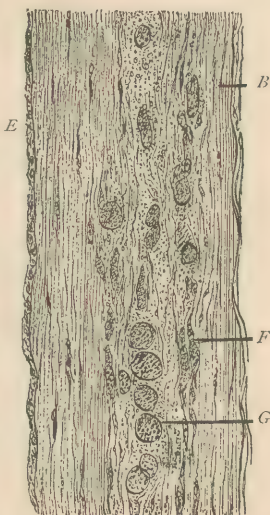


FIG. 6.—Acute Amnionitis. *E*, single epithelial cover; *B*, fibrous basis-substance; *F*, spindle-shaped protoplasmic bodies; *G*, inflammatory capsules proper.

Obviously this process was caused by the hemorrhage in the underlying placental tissue, the extravasated blood acting by mere pressure as an irritating agent.

With powers of six hundred diameters the attention is first fixed upon the villi, which are imbedded in, and surrounded by, either coagulated fibrin or compressed decidual tissue.

These villi are lacking the original myxomatous tissue altogether. In its stead we find a finely striated tissue, evidently fibrous connective tissue, with a varying number of partly branched, spindled protoplasmic bodies.

This tissue could not have arisen from compression of the original myxomatous tissue, but must have come after



FIG 7.—*E*, Covering epithelium; *F*, newly-formed fibrous connective tissue; *G*, spindle-shaped and globular protoplasmic bodies; *C*, clot of blood.

inflammatory processes that had led to the new formation of the fibrous tissue, and obliteration of the majority of the blood-vessels. It seems that some time elapsed between the advent of the hemorrhagic infarctus and the ultimate changes in the villi. In a few villi I have observed a large number of closely packed protoplasmic bodies, oblong and spindled, the only intimation of early formation of fibrous connective tissue. In a few places I have met with transverse sections of villi, which, instead of being myxomatous or fibrous, are composed of protoplasm with a few interspersed globular or oblong nuclei (see Fig. 4). Such places were found mainly at the junction of chorionic villi and decidua.

My studies in development of chorionic villi and placental villi cause me to make the suggestion that we have here to deal with protoplasmic buds of fully formed villi in a stage of indifference. I have observed that the villi grow by sending out protoplasmic masses, either sessile or pedunculated, starting from the epithelial cover of previously formed villi. Such buds are either made up of a uniformly granular protoplasm or of a multinuclear protoplasmic mass without any differentiation into epithelia, myxomatous tissue, or blood-vessels.

Since such protoplasmic buds were found surrounded by a dense fibrin of coagulum, it is reasonable to assume that this coagulum produced a wall around the buds, protecting them against compression or further changes. It is less likely that the protoplasmic buds penetrated into the coagulum. The decidual tissue, when present, is likewise characterized by the absence of the myxomatous reticulum and the presence of a delicate fibrous connective tissue, with varying number of oblong or spindled protoplasmic bodies (connective cells) imbedded in it. Very exceptionally we meet with a myxomatous reticulum composed of broad trabeculae with nuclei at the points of intersection.

The meshes of this reticulum show decided variation in size and contain a finely granular albuminous coagulum (see Fig. 5). Here a hyperplasia of the myxomatous reticulum seems to have developed, such as we sometimes observe in the lymphatics of the uterine mucosa in consequence of chronic endometritis.

Amnionitis.—The most striking features are seen in the inflammatory changes of the amnion. Being a non-vascular tissue it affords an excellent opportunity for the study of inflammatory tissue changes, almost as good as the much discussed cornea. Some pathologists consider tissue changes in inflammation a mooted question, looking

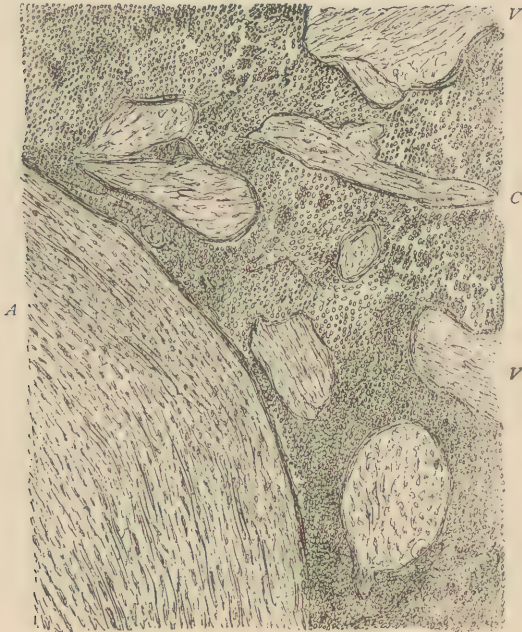


FIG 8.—Subacute Amnionitis. x 50. A, Enormously thickened amnion; V, V, villi of dense fibrous connective tissue; C, clots of blood.

upon inflammation as an emigration of colorless corpuscles in accordance with Cohnheim's theory. Of late this theory has lost ground, particularly in Germany. My specimens prove the presence of tissue changes with great certainty, a fact whose influence is in the direction of a broader definition of inflammation. If Cohnheim is right in saying that an aggregation of leucocytes is inflammation, from whence should we imagine protoplasmic bodies in the inflamed amnion to have sprung? There are no blood-



FIG. 9.—Acute Amnionitis. *R*, Rows of medullary corpuscles; *B*, slightly fibrous, basis-substance in process of reduction to protoplasm; *P*, accomplished reduction of basis-substance to protoplasm.

vessels, and all the blood-vessels of the subjacent placental tissue have either been compressed or destroyed by hemorrhage. The inflammation set in after this hemorrhage and there was no chance for the leucocytes to emigrate from destroyed blood-vessels. By Heitzmann and Stricker it is claimed that both the cells and the basis substance surrounding the cells are made up of protoplasm and are endowed with life. In inflammation a liquefaction of the basis substance takes place and the protoplasmic condition reappears, leading to an increase of the living matter and the appearance of inflammatory corpuscles that are identical with those embryonal or medullary corpuscles that have originally assisted in building up the tissues (see Fig. 6).

According to Stricker, there exist no migratory corpuscles in the corneal tissues, either in the normal or inflamed condition. What has been considered as such is, in reality, an alternating change of protoplasm to basis substance, and vice versa. My specimens of amnionitis show the different stages of this transformation, for we see, first, an increase in the bulbs of the connective tissue corpuscles, next the reappearance of the protoplasmic stage of the basis substance and the splitting up of the latter into inflammatory corpuscles of wide variation in size and shape.

An inflamed tissue is largely composed of medullary or inflammatory corpuscles, which remain united with one another so long as no suppuration has taken place. Separate inflammatory corpuscles are pus-corpuscles, and their sum total is an abscess. So long, however, as the inflammatory corpuscles remain connected the inflamed tissue remains a tissue, and is liable to return to the stage of basis-substance. Owing to the increase of living matter the bulk of the inflamed tissue is increased, and as such represents another termination of the inflammatory process,

i. e., hypertrophy, or, preferably, hyperplasia. This condition of the tissue is seen as a marked thickening of the amnion, being again fibrous in structure, though supplied with a larger number of protoplasmic bodies than normal (see Fig. 7).

The sequence of changes in the tissue under discussion was first hemorrhage from unknown causes, next irritation of the amnion and acute inflammation thereof, and at last thickening of the inflamed amnion in certain parts, under the form of hyperplasia. The same process is

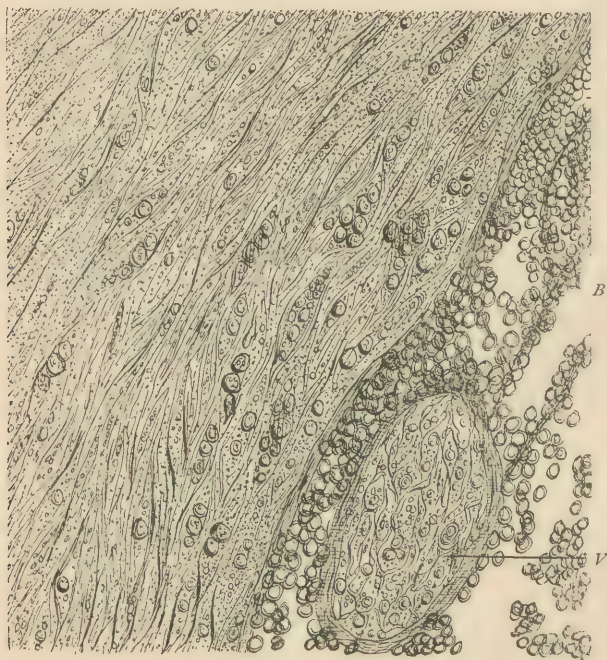


FIG. 10.—*A*, Hyperplastic amnion—dense fibrous connective tissue—protoplasmic bodies; *V*, hyperplastic fibrous tissue of a villus; *B*, red-blood corpuscles.

traceable in the remnants of decidual tissue and its villousities.

The transmutation of the original myxomatous into the final dense fibrous connective tissue was certainly not a direct one, but was through the intervening stage of reduction to the medullary condition, in other words, acute inflammation.

CASE III. *Subacute Amnionitis*.—The tissues examined in this were from a patient two months advanced in pregnancy, the placental tissues or residues having remained in the uterine cavity for six weeks following abortion. On account of metrorrhagia the placental shreds were removed by Dr. Wm. Hasslock of this city. They were placed in a half of one per cent. chromic acid solution and the specimens mounted in glycerine. The image under the microscope closely resembles that of Case II. We see enormous masses of blood with well-preserved red blood corpuscles, islands of previous villi of different sizes, and the whole surrounded by enormously thickened amnion, which partly again is traversed by hemorrhagic infarctions. With low powers of the microscope we recognize a condition of amnionitis and placentitis, more advanced even than in the previous case (see Fig. 8). Portions of the amniotic tissue are in a condition of acute inflammation, and since both the features of acute and chronic amnionitis are combined, it may best be termed subacute amnionitis. In order to illustrate the correctness of the views concerning acute inflammation as explained above, I have studied portions of the amnion in an acute inflammatory state, such as were seen best in the immediate vicinity of the blood-clot, with the highest available powers of the microscope, *i.e.*, twelve hundred diameters (see Fig. 9). We see protoplasmic bodies of varying sizes, partly single, partly in clusters and rows. In the latter situation the medullary corpuscles are mostly some-

what polyhedral, as if by mutual flattening. Such rows are most conspicuous where a certain amount of delicate fibrous tissue, *i.e.*, the basis substance of the amnion, is still preserved. That medullary corpuscles may appear in the middle of the basis substance is plainly shown in the right upper corner of the drawing, in the field marked B. The fact is inexplicable, unless on the ground that the basis substance itself is viable. Life, however, is not in the basis substance at large, for we know that there is a large amount of glue-yielding substance which, as such, cannot be the seat of life. This property seems inherent only in the part of the basis substance that is traversed by an extremely delicate reticulum.

Should the glue-yielding part of the basis substance become fluid by its disintegration or liquefaction, the immediate result is the re-establishment of protoplasm, in which the reticulum of living matter is seen plainer than in the basis-substance, as demonstrated in the left lower corner of the drawing at P. Here we notice mostly scattered lumps of protoplasm, greatly differing in size, from a small, almost homogeneous lump up to a nucleated medullary corpuscle.

The main mass of both the amnion and the decidual tissue is in a condition that we know as hyperplasia. In this condition we see a dense fibrous connective tissue, between the bundles of which there are numerous globular bodies, the so-called connective-tissue corpuscles, in rows or clusters.

These are the remnants of the inflammatory corpuscles which, by becoming spindled, produce the basis substance (see Fig. 10). Amnion is an avascular tissue and, even in a chronic inflammation in hyperplasia, will not show any traces of blood-vessels, contrary to the cornea, which in the same condition is invariably supplied with blood-vessels growing from the periphery toward the centre. The

villi likewise exhibit occasional stages of acute placentitis, though their majority is made up of a more or less conspicuous fibrous connective tissue, with an almost complete loss of capillaries. Unquestionably there must have been left a vascular connection with the decidua, for otherwise we could not understand the inflammatory changes of the placenta and amnion.

Instead of becoming necrotic or absorbed, as is the case with tissue-shreds, isolated by hemorrhage, in this case important tissue-changes have taken place which go far toward proving that nutrition of the placenta had not been entirely destroyed.

I wish to here record my indebtedness to Dr. Carl Heitzmann for the use of his laboratory, and especially for many valuable suggestions afforded during my investigations.

151 East 34th Street.

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VULVAR OEDEMA COMPLICATING PREGNANCY.

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OEDEMA of the vulva, sufficient to entirely occlude the vaginal outlet, with eclampsia threatening to supervene at any moment, invites a report of the following case.

M. L., twenty-two years old, Irish, domestic, primipara, in the last fortnight of pregnancy, was admitted to the Lying-in Department of the New York Polyclinic Hospital, February 6, 1890. Her health had been excellent previous to her pregnancy. Nothing indicative of kidney disease had been observed.



On physical examination the lower limbs were found highly oedematous. The right foot measured around the

instep 24.1 cm. The left foot 26.7 cm. The right ankle measured 28.3 cm. and the left ankle 33.3 cm. The skin at the most distended portion was much thinned and apparently at the limit of distension. The œdema extended to the knees. The vulva was enormously swollen, as shown in the cut. The right half received the greater part of the infiltrated serum, causing the labium to bulge over and occlude the vaginal orifice, the left labium being almost entirely concealed. The infiltration was extending to the left labium and buttock. The labial swelling on the right side measured, in the anterior median line, 18.1 cm., in the transverse median line 16.3 cm. and in the entire circumference 31.1 cm. At the lower portion of the swelling was a pin-hole opening through which pus was exuding. The limit of distension without rupture was here reached, the skin being quite translucent.

Abdominal palpation revealed a characteristic primiparous condition.

The abdominal wall was firm, tending to oppose free expansion and elevation of the uterus. The latter was firm, hard and of average size. The fœtus was lying with the occiput extended to the left transverse. The amniotic fluid being scant, foetal movement was limited. The head rested very firmly in the pelvic inlet. The urine was strongly acid, odorless, of syrupy consistence, turbid and of yellow color. Specific gravity 1027, quantity passed during the first 24 hours 37½ ounces. There was a flaky whitish sediment. Albumen to the amount of $\frac{1}{10}$ per cent. was present. There were granular casts, a few hyaline casts and an abundance of pus cells, the latter probably coming from the tumor. Perspiration was profuse.

The patient suffered from vertigo, frontal headache, occasional flashes of light before the eyes, dyspepsia, nausea and vomiting. She stated that the œdema in the lower limbs first appeared two weeks before and gradually in-

creased in amount. The labial swelling began three days before and became severe in twenty-four hours. It was due, as she thought, to her being obliged to work hard standing all day.

The group of symptoms present in this patient pointed emphatically to the imminence of eclampsia. She was given a general warm bath, followed by an enema of one ounce of sulphate of magnesia. The foot of her bed was elevated and pillows were placed under her legs. The external genitalia were washed with a 1-3000 corrosive sublimate solution.

She was then given *per os* $\frac{1}{100}$ of a grain of nitro-glycerine every three hours. The bowels moved freely. Within the twenty-four hours following the administration of nitro-glycerine 67 ounces of urine were passed, an increase of $29\frac{1}{2}$ ounces over the preceding twenty-four hours.

Albumen diminished to $\frac{1}{24}$ per cent., a relative and actual reduction. The œdema diminished slightly in the right limb, remained stationary in the left limb, and increased in the vulva by extension to the left labium and buttock.

Numerous punctures under antiseptic precautions were made in the inferior portion of the right labium, the fluid squirting out in little streams at first.

The swollen parts were covered with gauze soaked in sublimate solution and a folded blanket laid on top to secure gentle and continuous discharge of the serum. In twelve hours the vulvar swelling had almost entirely disappeared. Twelve hours later I undertook, in the presence of the obstetric class, external podalic version with the object of removing the head pressure from the pelvic inlet, believing that most of the unfavorable conditions present were caused by the impacted head interfering with the pelvic and renal circulation. I did not object to precipitating labor, the time for the confinement having arrived. I

employed Wigand's method in the manner practised by me in a series of cases reported in *The Medical Record* of May 26, 1888, but found the mobility of the foetus so slight that the head could not be disengaged with reasonable effort, and ceased further attempts. Labor ensued in ten hours, perhaps, excited by my manipulation. After a normal labor the patient experienced no further difficulty, all the abnormal conditions gradually disappearing.

An interesting question arose in this case. Had eclampsia supervened before the reduction of the vulvar swelling was accomplished, would not a Cæsarean section have offered greater safety than a vaginal delivery which involved from twelve to twenty-four hours for the reduction of the tumor, the excitation of labor and cervical dilation and delivery.

This question did arise for eclampsia seemed probable and was decided in favor of abdominal section for the following reasons: the mortality in cases of Cæsarean section rests to-day at 27.52 per cent. in the total results of all countries and, better still, only 15 per cent. in Germany.*

On the other hand, the mortality from eclampsia *beginning before labor* is given by Galabin † as 50 per cent. and Lohlein ‡ as 40.05 per cent.

Eclampsia is generally the product of temporary functional or mechanical derangement of the circulatory or eliminative apparatus or both. That Cæsarean section performed before the convulsions of eclampsia could have caused much cyanosis or coma, would exert a favorable influence in restraining the spasms is reasonable. That at

* Dr. Harris, in the *American System of Obstetrics*, Vol. II, page 260, records 80 cases in Germany, with 12 deaths and 38 operators; and a total of 108 recoveries in 149 cases in the hands of 82 operators in various countries.

† In the *American System of Obstetrics*, Vol. II, page 76, Galabin records the mortality of cases arising before labor as 50 per cent., during labor as 25 per cent., and after labor as 8 per cent.

‡ In *Lusk's Obstetrics*, page 564, Löhlein gives the mortality as 40.05 per cent. before and 6.66 per cent. after labor.

this stage the eclampsia would not have produced conditions unfavorable to a Cæsarean section is likewise reasonable. Through the courtesy of Dr. R. P. Harris, I learn that three Cæsarean sections have been performed for eclampsia. The first done under the old method, in 1878, proved fatal in 73 hours. The other two were by Prof. Halbertsma, who employed Saenger's method and resulted in saving both mothers and children. My patient presented more favorable conditions than the average for a successful Cæsarean section, and much more unfavorable than the average in eclampsia arising before labor, had it occurred with the cervix beyond our reach for twelve hours. With a statistical record of at least two deaths from eclampsia to one from Cæsarean section, the latter certainly seems the more conservative procedure.

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VERSION BEFORE LABOR IN MALPOSITIONS OF THE FŒTUS.

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FŒTAL mortality, maternal lacerations of the cervix and perineum, vesico-vaginal and recto-vaginal fistulæ, uterine ruptures, and pelvic inflammations are so much less frequent, proportionally, in occipito-anterior presentations in labor than in any others, that any procedure that will convert the latter into occipito-anterior presentations without adding new dangers should receive hearty approbation. In occipito-posterior presentations a severe laceration of the perineum, and perhaps the sphincter ani, is the rule where craniotomy is not performed, unless episiotomy be judiciously employed. In addition, the dangers of prolonged and laborious labor fall to the mother's lot.

In breach presentations, the necessity of haste in the completion of the second stage imposes great risk to the cervix and perineum. In transverse presentations the dangers to the mother are very great. Spontaneous version or evolution, artificial version or evisceration, are means of relief that are usually obtained only after prolonged exhaustion and pressure, with local blood stasis, have supervened. Generally speaking, the only cases in which occipital presentations may be undesirable are such as can be accomplished by podalic version in preference to cephalic perforation.

We have, then, an argument of almost universal inclusion in favor of the conversion of all presentations to that of occipito-anterior, depending upon the comparative dangers involved in bringing about such changes. Version, when the cervix has dilated and the membranes ruptured, is a difficult and dangerous operation, on account of the active contractions of the uterus, the retraction that has taken

place, with loss of space in the uterus, and the risk of sepsis. Wigand, in 1807, argued clearly, forcibly, and effectually in favor of version before labor, and with such complete monographs in our possession to-day as those of Pinard on "Abdominal Palpation," and Mundé on "Obstetrical Palpation," it seems strange that this procedure is not more generally employed.

In version before dilatation of the cervix the conditions that will increase the difficulties are: Scarcity of amniotic fluid, largeness of the child, thickness of the abdominal wall, and firmness or irritability of the uterus. But it is in reverse conditions, *i.e.*, abundance of amniotic fluid, diminutiveness of the fœtus, and laxity of the uterine and abdominal walls, that breech and transverse presentations usually occur. In fact they are frequently the result of such conditions. Therefore, as a rule, the conditions are favorable for turning before labor in those presentations that require correction.

If we hesitate in completing version before labor where the accomplishment is difficult—a situation most apt to exist in breech presentations—we must consider which will be more conservative to both mother and child, the difficult immediate version, or the final delivery in the malposition. Immediate version should not always gain our approval. The practical application of early version involves a change of custom with both patient and physician, probably a bigger stumbling-block than the operation itself. Patients must be trained to recognize the early engagement of the accoucheur, and abdominal examinations before labor as the proper custom. Inasmuch as vaginal examinations are usually unnecessary in making a diagnosis of foetal position in utero, patients will more readily submit to investigation. I have found the following general rules most useful and necessary in making the diagnosis of foetal position by abdominal palpation.

With the patient lying upon her back, the thighs well flexed, and bladder empty, we should first seek the foetal head. If it is at the "brim," by passing the fingers of both hands downward and backward, starting two inches above the symphysis, with a space of three inches between the index-fingers, it will be felt as a hard, incompressible body of a diameter corresponding to that of the foetal head. In most cases, after the eighth month, the portion of the head lowest in the pelvis will be beyond the fingers' reach. If the head is not at the brim, search for it in other parts until found. It is much the easiest recognized of any of the foetal parts. If it is at the fundus or at either side it will, in cases of difficulty due to excess of amniotic fluid, be found more readily by abdominal *ballottement*. The breech very rarely affords this sign. The hard character of the head, as compared with the fleshy shoulder, back, or breech, will also be recognized in this class of cases, and also when the abdominal walls are thick.

If the walls are thick and the amniotic fluid scarce, cephalic outlines or density can almost invariably be recognized. Having found the head, the back should next be found, not the breech. Following the head there will be found either a large cylindrical body, the back; or small irregular bodies, the extremities. Frequently both can be felt. In such cases, the foetus lies upon one side. Knowing which way the back is turned, we can tell the position of the head or breech at the pelvic inlet, unless it be a transverse presentation. If the back lies forward in cephalic presentations, the occiput will be anterior and right or left, as the back lies to the right or left; or if it is a face presentation, a fact next to be determined, the diagnosis will not be difficult. The position of the back will, in the same way, give us the position of the foetal sacrum in breech presentations. To determine the degree of extension or flexion of the occiput, a fact necessary for diagnosing by ab-

dominal palpation, face, brow, or occipital presentations, ascertain the angle of junction of the head with the shoulders and back. If the angle is very obtuse and unrecognizable, it indicates extension of the occiput ; if acute, extension of the chin.

Palpation through the anterior wall of the cervix with the index-finger will serve to confirm judgment as to the *presentation*, but not as to the *position* of the presenting part. The custom in serious cases of introducing the entire hand to diagnosticate "position" cannot become obsolete too soon. The differentiation between the breech and head at the inlet is gained in this way : In head presentations the fingers cannot reach the most inferior portion, as the head rests too low in the pelvis, except in hydrocephalus or pelvic distortion ; but in breech presentations the fingers can generally pass below the foetus and meet with only the maternal tissues between them.

Before attempting version, localization of the placenta would be desirable, but the "bruit" is a diagnostic myth, and recognition of its additional thickness to the uterine wall too difficult. We can only be careful in manipulating the fundus. When to perform version before labor will depend upon individual conditions. The foetus in many women examined at the seventh month will be found with the breech presenting, but is born at full term with the occiput first.

The proportion of breech and transverse cases is much greater in premature than in full-term labors. But version just before labor is more difficult than it is several weeks earlier. If the amniotic fluid remains relatively large in amount up to the end of pregnancy, then version should be delayed to two weeks before labor, and *vice versa* ; if the child is getting large enough to occupy so much space that turning will soon be difficult, then immediate version should be practised.

The possible dangers in version before labor are in the order given : Laceration of the amniotic membranes, of the placenta, cord, uterus, and indirectly from any of these, death of the fœtus ; labor may be precipitated. None of these dangers are at all imminent. In performing version, if the abdominal walls are tense from intestinal overloading, a half-drachm to a drachm of fl. ext. cascara sagrada will, by emptying the bowels, give more latitude in turning. Emptying of the bladder should never be omitted.

Gentleness, warm hands, cessation of effort during uterine contractions, and absence of all haste, will command success where opposite methods will cause failure and perhaps lacerations of the uterine contents. Anæsthesia is uncalled for. In converting a breech presentation into a cephalic, it will be found more difficult to pass the breech up out of the cervical segment than to bring the cephalic end down to the transverse diameter of the uterus, especially when the cervix rests well down in the pelvis. It may then be found necessary to elevate the intra-vaginal portion.

If it is a left sacro-anterior, the left finger should be introduced, and pressure made upward and to the mother's left, and *vice versa* in a right sacro-anterior. In swinging the long diameter of the fœtus beyond the transverse diameter of the uterus, an assistant may prove useful by making pressure upon the fundus and cervix, with the object of increasing the transverse diameter. In sacro-posterior positions, rotation of the fœtal back to the front should be undertaken, before attempting cephalic version, as it is easier to do in a breech presentation. If much difficulty presents in trying to lift the breech out of the pelvis, place the patient in the knee-chest position, and admit air into the vagina, thus permitting the uterus to fall away from the pelvic inlet. This aids in displacing the presenting part from out the inlet. There is a tendency in transverse positions for the head to glide to the fundus in the genu-pectoral position.

Rotation of the foetal back to the front is difficult, from a want of leverage. The lower limbs afford the best purchase in rotation on the foetal axis. If the foetal back lies to the right posterior side of the uterus, the knees should be pressed downward and backward to the left posterior side. Then, while they are held here with one hand, pressure with the other should be made from as posterior a part as possible on the right side against the foetal back, and effort directed to lifting it forward and thus over to the left. In performing version before labor we must, to avoid injuries, exercise extreme care not to use one point for pressure too long, or use too great force in displacing the foetus. When labor has begun, these considerations are of less importance. Version accomplished, if before labor, there is no guarantee that spontaneous reversion to the previous malposition may not ensue. A study of the various causes of the malpositions in each case will aid in directing treatment for retaining the foetus in place.

In hydrocephalic cases, the condition being recognized, if at all, by manual estimate through the abdominal walls, of the disproportionate size of the head, the frequency of breech presentations is said to be due to the decreased specific gravity of the head permitting the breech to gravitate to the pelvis. Such a cause could scarcely act late in pregnancy, so that version would prove lasting, except for the fact that here, as in contracted pelves, impaction of the head in the inlet is difficult, and much mobility prevails in the presenting part, shoulder presentations are apt to result. Not very much can be done to insure fixation. Mild compression of the lateral abdominal walls, by means of padded splints or bandages, will prove useful. In malpositions due to laxity in the uterine and abdominal walls, a supporter or bandage, such as that recommended by Pinard, which supports the entire abdomen, and prevents sagging of the fundus, is needed. In hydramnios, where extreme disten-

tion of the uterus exists, bandaging is useless. But it is especially desirable in hydramnios to have an occipital presentation, as the child's vitality is poor, the uterus sluggish and weak, causing delay in breech cases that is very fatal to the child. Having obtained the desired fœtal position the dangers of a bad result justify, if the pregnancy is within a couple of weeks of full term, artificial precipitation of labor.

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No. of Case.	No. of Pregnancy.	Period of Pregnancy	Position of Fœtus.	Etiology.	Version.	Method.	Presentation when delivered.	Remarks.
1	Third	11 days before labor at full term.	Right sacro-anterior.	Unknown	Cephalic to left occipital anterior.	Wigand's method. Both hands external.	Left occipital anterior.	First labor was a breech.
2	Second	12 days before labor at full term.	Right sacro-anterior.	Short conjugate diameter.	Cephalic to left occipital anterior.	Wigand's method.	Left occipital anterior.	Lateral compresses were used for three days after version.
3	Second	In labor at full term.	Right mento-iliac.	Chest very large. Over 3 gallons of amniotic fluid.	Left occipital anterior.	Schatz's method.	Left occipital anterior.	Head had to be held in place until forceps could be applied and traction enough given to get impaction of occiput, then forceps were removed. Child weighed 13½ pounds.
4	Fourth	5 days before labor at 8½ months.	Dorso anterior. Right shoulder.	Small child. Uterus very much relaxed.	Left occipital anterior.	Wigand's method.	Fetus had re-turned to former position. Changed to left occipital anterior by Wright's method.	Child weighed 6¾ pounds.
5	First	In labor at full term.	Right sacro-anterior.	Unknown	Left occipital anterior.	Knee-chest position until the breech was displaced from the inlet; then dorsal position. Wigand's method.	Left occipital anterior.	Membranes ruptured unintentionally, but this did not prevent external version.
6	Ninth	2 weeks before labor at full term.	Transverse dorso-anterior. Right shoulder to left of inlet.	Unknown	Left occipital anterior attempted and given up.	Wigand's method.	Uterus was irritable, and fœtus rather immovable. Thought best to wait.
		Second trial in labor at full term.	Right shoulder presenting, dorso anterior.	Left occipital anterior.	Wigand's method.	Left occipital anterior.	Head had to be held in place until fixed in pelvis by dilatation of os and rupture of membranes.

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